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REPORT DOCUMENTATION PAGE			Form Approved OMB No. 0704-0188	
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1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE 24.May.05	3. REPORT TYPE AND DATES COVERED MAJOR REPORT		
4. TITLE AND SUBTITLE OPERATING ROOM TELEPHONE MICROBIAL FLORA		5. FUNDING NUMBERS		
6. AUTHOR(S) CAPT SHINN ANTOINETTE M				
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) UNIFORMED SERVICES UNIV OF HEALTH SCIENC		8. PERFORMING ORGANIZATION REPORT NUMBER CI04-1093		
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES) THE DEPARTMENT OF THE AIR FORCE AFIT/CIA, BLDG 125 2950 P STREET WPAFB OH 45433		10. SPONSORING/MONITORING AGENCY REPORT NUMBER		
11. SUPPLEMENTARY NOTES				
12a. DISTRIBUTION AVAILABILITY STATEMENT Unlimited distribution In Accordance With AFI 35-205/AFIT Sup 1		12b. DISTRIBUTION CODE		
13. ABSTRACT (Maximum 200 words)				
<p>DISTRIBUTION STATEMENT A Approved for Public Release Distribution Unlimited</p> <p>20050602 007</p>				
14. SUBJECT TERMS			15. NUMBER OF PAGES 26	
			16. PRICE CODE	
17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT	

Running head: TELEPHONES AS FOMITES

Operating Room Telephone Microbial Flora

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March 31, 2005

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Abstract

Introduction: There are approximately 500,000 surgical site infections per year in the United States [1]. The purpose of this study was to determine if the bacteria most frequently involved in Surgical Site Infections (SSI) could be found on telephones in the Operating Room (OR).

Methods: A total of 26 cultures were taken from telephones within 14 operating rooms and two sub-sterile rooms at a large, teaching, medical center. Bacteria were identified using standard laboratory procedures and the Vitek system version 7.02. *Results:* Of the 52 isolates discovered, the following bacteria were identified: *Acinetobacter calcoaceticus-baumannii* complex 1.9%, *Pseudomonas aeruginosa* 1.9%, *Agrobacterium radiobacter/tumefaciens* 1.9%, Coagulase-negative *Staphylococcus* 82.7%, *Micrococcus* 3.8 %, *Streptococcus* non-group D 5.8%, and one unidentified gram-negative rod 1.9%.

Introduction

There are approximately 500,000 surgical site infections per year in the United States [1]. Nosocomial infections contribute to prolonged antimicrobial treatments, length-of-stays, and even death. The Centers for Disease Control (CDC) reports that in 1999 the most prevalent causes of Surgical Site Infections (SSI) were: *Staphylococcus aureus* (*S. aureus*), Coagulase-negative *Staphylococci* (CNS), *Enterococcus species* (*spp*), and *Escherichia coli* (*E. coli*) [2] [3]. There have been no published changes to the prevalence of these bacteria in relation to SSI since 1999. In addition, a study published in 2003 reports that extremes of costs for SSIs are as high as \$92,363 for patients with Methicillin-resistant *Staphylococcus aureus* (MRSA) SSI[4].

The most common source of SSI is endogenous floras [5], but exogenous floras are also a possible cause of SSI [3] [6]. If exogenous floras are causing some surgical site infections, how are they being transmitted? Could the hands of healthcare workers be a source? What other surfaces might be involved via direct or indirect contact with patients? One inanimate item in the operating room (OR) frequently contacted by the hands of staff is the telephone. Could telephones in the OR serve as a source of surgical site infections? An inanimate surface that is implicated in a nosocomial infection is termed as a fomite. Are telephones in the OR fomites? Given the potential impact of nosocomial infections in the perioperative setting, research is needed to describe if the bacteria most frequently involved in SSIs can be found on telephones in the OR. The purpose of this paper is to describe a study we conducted to identify and quantify bacterial contamination on telephones in the OR of a large teaching medical center.

Literature Review

Before we can address the questions raised above, we must understand the many factors associated with nosocomial infections. The chain of infection model provides the best framework for depicting the relationship among these factors and SSIs. Our literature review includes a thorough explanation of the chain of infection model, the relationship among these factors, and a discussion of the current literature on environmental surfaces as fomites.

According to the chain of infection model, a causative agent or pathogen survives within a reservoir, exits the reservoir via a mode of transmission, and enters a susceptible host, thereby, causing disease [7]. Intervention in any part of this process can stop the transmission of disease. The reservoir can include plants, animals, soil, water, and inanimate surfaces [8]. Of these, the most likely exogenous reservoir in the surgical setting is either human or an inanimate surface. Both reservoirs are capable of becoming transmission agents.

Inanimate Surfaces as Reservoirs

The evaluation of inanimate surfaces is best categorized by Spaulding's Classification System. Within this system, items are classified as: Critical, Semi-critical, and Non-critical [9]. Critical items present a significant risk of infection if microorganisms are present because these items come in contact with sterile tissues. Semi-critical items pose less risk, because they are in contact with mucous membranes or non-intact skin. Non-critical items are only in contact with intact skin and pose little risk of infection. However, Non-critical items used in patient care can serve as a mode of secondary transmission by providing a reservoir that can contaminate the hands of healthcare workers [10]. This mode of transmission, surface to hand transfer of bacteria, is well documented in the literature [11] [12].

Animate Reservoirs as Transfer Agent

Proper hand washing is known as one of the most important steps in preventing infections [9]. Despite several studies documenting hands as carriers of infection [13] [14], hand washing compliance has been shown to be as low as 9% for medical intensive care unit (ICU) health care workers and 3% for cardiac surgery ICU health care workers [15]. More to the point, as few as 58% of anesthesiologists report that they wash their hands after contact with every patient [16] and compliance with hand-cleansing in a post-anesthesia care unit was shown to be 12.5% [17]. If proper hand hygiene is not exercised, items frequently contacted by hands could serve as reservoirs and those reservoirs could further serve to contaminate hands; thereby increasing the chance of spreading infections to patients during hand-to-patient contact.

Environmental Surfaces as Fomites

Even though the importance of cleaning environmental surfaces is well recognized as a standard of care, there is little research available which describes the relationship between the quantity of pathogens present on surfaces and increased nosocomial infection rates [18]. Bacteria are capable of transferring antibiotic resistance [19] [20] [21] [22] [23], therefore it can be argued that where bacteria are allowed to survive on environmental surfaces, antibiotic resistance could be transferred. It is reasonable to question the cleanliness of environmental surfaces in the surgical setting, especially when as much as 32% of anesthesia equipment has been found to have occult blood present [24].

The role of inanimate surfaces as fomites is not well documented. Some recent studies suggest there is no link between infection rates and surface contamination [25] [26] [27] while other studies demonstrate that environmental surfaces and nursing uniforms have increased contamination from patients known to be infected or colonized with MRSA [28]. A wide range

of environmental surfaces have been shown to be sources of nosocomial infection, including an electronic ear probe [29], a stretcher frame, a shower handle [28], and operating room surfaces [30]. In order to determine the likelihood of bacterial presence on telephones and subsequent transfer via hands, we reviewed the literature to identify survival times of the bacteria most frequently implicated in surgical site infections on hands and inanimate surfaces. These bacteria are: *S. aureus*, CNS, *Enterococcus spp*, and *E. coli*. Our literature review also included MRSA and Vancomycin-resistant *Enterococcus (VRE)* as both of these bacteria are variants of *S. aureus* and *Enterococcus spp*. Due to the lack of studies specific to telephone surfaces, we also used plastic surfaces as a substitute search criteria for telephones.

Table 1 is a compilation of our literature review and is reflective of experimental and quasi-experimental studies dating back to 1989. While some of the studies are dated, in many cases these studies are either landmark or sole source references. Each of the studies presented in Table 1 used different inoculum concentrations and techniques, and provided us with evidence that bacteria might be present on OR telephones. With this evidence we reviewed the literature on studies involving telephones as a reservoir for bacteria and concluded that time alone would not eliminate bacteria sufficiently. The importance of hand washing, aseptic technique, and surface decontamination was evident.

{Please insert “Table 1 Bacterial Survival on Surfaces”} [30] [31] [32] [33] [34] [35] [36]

In one study conducted to document environmental surfaces and hands of healthcare workers as reservoirs, consisting of 26 telephone cultures in an ICU, the researchers found *S. aureus*, *Acinetobacter calcoaceticus (A. calcoaceticus)*, and *Pseudomonas spp* [34]. In another study of Non-critical items frequently in contact with hands of staff in the hospital, 20 telephones from the OR, ICU, Recovery, and emergency room (ER) were cultured and resulted in no gram-

negative bacteria being identified [37]. Cozanitis, Grant, & Makela (1978) cultured 11 telephones in an ICU and identified CNS, Coagulase-positive *Staphylococcus*, gram-positive rods and alpha-hemolytic *Streptococcus* [38]. Lastly, a study conducted to identify the bacteria on public telephone hand-pieces at a high school showed increasing numbers of bacteria on telephones from morning to afternoon with CNS being the predominant bacteria identified [39].

The literature review has shown the potential for bacteria to be present on telephones for variable lengths of time and has demonstrated that there is frequently a lack of hand washing and decontamination of environmental surfaces by hospital staff. Additionally, inanimate surfaces have been implicated in infections. In a study conducted by Rusin, Maxwell, and Gerba (2002) a link is clearly created between the transfer of bacteria from telephones to hands and from hands to other skin surfaces. Rusin et al. demonstrated that *Micrococcus luteus* (*M. luteus*) can be transferred from telephones to hands with approximately 41% efficiency and from hands to the mouth at the same rate of 41% [40].

Methods

Design

The purpose of this descriptive study was to determine if the bacteria most frequently involved in SSI could be found on telephones in the OR of a large teaching medical center. We focused exclusively on *S. aureus*, CNS, *Enterococcus spp*, *E. coli*, MRSA and VRE. We developed an algorithm (see figure 1) and written guidelines for our data collection and analysis procedures. Data were collected by swab and culture techniques to make observations about bacteria colony counts. A data collection sheet, was designed based on our algorithm and was utilized to capture data related to: surgical service, the time of day when cultures were collected, location of the telephone in the perioperative environment, surgical case number, and OR temperature and OR humidity at the time of sampling. Our research protocol was approved by the Institutional Review Boards of the medical center and the Uniformed Services University of Health Sciences.

Sample and Setting

We used a quota based convenience sample of 30 cultures from telephones within the ORs at the medical center. Twenty-six cultures were obtained from telephones within 14 operating rooms and two sub-sterile rooms. Two control cultures and two double cultures were also collected. Specimen collection was divided between two days that were separated by 19 days in order to decrease the likelihood that perioperative personnel might alter hand washing, aseptic techniques, and environmental disinfection, because they were aware of our study and data collection [41].

Specimen Collection and Analysis Procedures

In order to ensure precision in the collection of data, we (the principal investigator and

two co-investigators oriented to the medical center's lab and became familiar with testing supplies and procedures. With the assistance of our Microbiologist Associate Investigator, we developed a guideline for all testing procedures, based on the algorithm in figure 1. The guideline was incorporated into a standardized specimen data collection and analysis sheet for use in recording the identification and interpretation of bacteria. Laboratory personnel evaluated our skills with a practical exam utilizing eight known bacterial isolates.

Cultures were taken from OR telephones at the end of surgical cases to eliminate unnecessary traffic through the surgical area and prior to staff cleaning of the surgical suites. Cultures were taken in the same manner by all three investigators wearing sterile gloves. All four sides of the telephone hand piece handles were swabbed. The posterior swab path included one vertical pass from earpiece to mouthpiece while holding the swab on its side and rotating or rolling it across the surface. Remel Bacti-Swabs with non-nutritive modified Stuart's Medium were used to obtain bacterial sampling. Validity and reliability testing was obtained by random double culturing and random control culturing techniques. The double culture technique included the swabbing of telephones in the manner described previously with two consecutive culturettes attempting to eliminate path over-run. These culturettes were labeled so that one investigator was blinded to the source. One control culture was randomly selected for each culture batch. The control culturettes were opened and re-sealed without exposure to contaminants.

After sampling, the swabs were returned aseptically to their cases, labeled and numbered sequentially. Swabs were carried to the lab within 15 minutes of sampling. All samples were streaked for isolation onto tripticase soy agar with 5% sheep blood agar (Remel, reference # 01202), chocolate agar (Remel, reference # 01302), and MacConkey agar (Remel reference #

01552), respectively. The agar plates were incubated at 35 degrees Celsius (°C) for 24 hours. Chocolate and blood agar plates were incubated in 4% carbon dioxide (CO₂) while MacConkey agar plates were incubated in 1.2 % CO₂. After the first 24 hours, the entire bacterial floras were visually quantified into the number of colonies present. If no colonies were present at 24 hours, confirmation was performed at 48 hours.

Bacteria were initially identified based on growth within the medium. Bacterial growth on MacConkey was considered gram-negative and was tested to rule out *Acinetobacter spp.*, *Pseudomonas aeruginosa* (*P. aeruginosa*), *Haemophilus influenza* (*H. influenza*) and *Neisseria gonorrhea* (*N. gonorrhea*). Bacterial growth on chocolate plates was used to identify *H. influenza* and *N. gonorrhea*, which were ruled out based on gram stain morphology and microscopic examination. Bacterial growth on blood agar was used to support the growth and evaluation of *S. aureus*, MRSA, CNS, *Enterococcus spp.*, VRE, and *E. coli*.

Bacteria were then identified by: 1) shape- spherical (coccus), rod-like (bacillus), or spiral (spirochete); and 2) cell wall- gram-positive or gram-negative as seen with gram stain. Gram-negative rods were tested for oxidase (Remel, ref #425506) and indole (Remel, ref #21245) reaction. Positive oxidase and negative indole results ruled out *E. coli* and were tested further to rule out *A. calcoaceticus-baumannii* and *P. aeruginosa*. Gram-positive bacteria were initially tested using 3% hydrogen peroxide for catalase testing, which was used to differentiate group 1 (*Micrococcus*, CNS, *S. aureus*, and MRSA) from group 2 bacteria (*Enterococcus spp.* and other *Streptococcaceae*). Group 1 bacteria were then tested with Remel Staphaurex Plus (ref # 30950102) to rule in *S. aureus*. Bacteria that were negative for Staphaurex Plus were then tested with the Microdase test (Remel, ref # 21132) to differentiate CNS from *Micrococcus spp.* Catalase negative bacteria were analyzed using the Boule Phadebact D test to differentiate

potential *Enterococcus* from other *Streptococcus* spp.

The Vitek system version 7.02 was used with BioMerieux Gram Positive Identification (GPI) and Gram-negative Identification + (GNI+) cards to identify *A. calcoaceticus-baumannii* complex, *Agrobacterium radiobacter/tumefaciens* (*A. radiobacter/tumefaciens*), and 9 of the 43 isolates of CNS. A single double culture of CNS was also analyzed via GPI card. Identification of *P. aeruginosa* was based on the following test results: gram-negative rod, oxidase positive, catalase positive, presence of motility, and growth at 42°C in tripticase soy broth (TSB). The methods described above are in compliance with standard culture technique [42] [43].

The counting of colonies was performed by two investigators individually and digital photos were taken. Additionally, the surface area of the four vertical swabbing paths was calculated to determine Colony Forming Units (CFU)/centimeter squared (cm²). The maximum swab path width was measured at 3 mm. The length or distance of this path was measured at 95.6 cm. To find the surface area in cm², the length (95.6 cm) was multiplied by width (0.3 cm) for a total of 28.7 cm², which is nearly equivalent to the surface area of a RODAC agar plate with a 6 cm diameter ($3.14 \times 3^2 = 28.26 \text{ cm}^2$). Data were entered into a spreadsheet by two investigators individually, using the completed specimen data collection and analysis sheet. A test of inter-rater reliability revealed 100% agreement between the two investigators on all data entered into the two separate spreadsheets.

Laboratory and Equipment

The lab we utilized was accredited by the Commission of Laboratory Accreditation of the College of American Pathologists (CAP) in 2004. The reliability and validity of the Vitek system is well established among medical laboratories. Quality Controls (QC) were conducted on all identification card lots used in this study. Digital photographs were taken using an

Olympus D-380 camera with 2.0 mega pixels effect and five times digital zoom.

Proficiency testing for the Vitek was performed three times during the year of our study. At the time of preparation of this paper, test results were only available for 2 of the 3 proficiency tests; these proficiency tests showed $\geq 86\%$ accuracy of bacterial identification and $\geq 92\%$ performance satisfaction with 100% antigen detection. This level of testing is in accordance with CAP accreditation. The Vitek system is approved by the Food and Drug Administration (FDA) for both gram-positive and gram-negative bacterial identification and sensitivity testing (1991 & 1996).

Phenotypic Testing Agents

We used several phenotypic testing agents while conducting our study. None of the agents were used beyond their expiration date. We used the same lot numbers among agar plates, culturesses, Vitek cards and all other supplies. The only exception to this was the Microdase test, which did change lot numbers during our second batch of testing. We present a brief literature review of our testing agents here to demonstrate reliability and validity.

Staphaurex Plus in comparison with tube Coagulase test on *S. aureus* isolates has been found to have a relative sensitivity of 99.4%, a relative specificity of 95.5% and an overall agreement of 98.4% [44]. In another study, Staphaurex Plus was found to be 99.6% sensitive and 93.9% specific [45]. During 510K testing on the Vitek GNI+ card, a Centers for Disease Control (CDC) gram-negative challenge set was utilized. Gram-negative bacteria were correctly identified to the species level 85.2% (77.4% to 91.1%) of the time. Correct identifications to the genus level occurred at 88.7% (81.4% to 93.8%). Misidentifications occurred at 7.8% (3.6% to 14.3%) and no identifications at 7% (3.1% to 13.2%) [46]. The Microdase test we utilized is described as "...the simplest and most rapid methods for separating *Staphylococci* from

Micrococci”[47]. In another evaluation of the Microdase test it was described as “the oxidase test (Microdase disk; Remel) proved to be the most sensitive (100%) and was sufficiently specific (99%) for providing a rapid means of accurately differentiating between *Staphylococci* and *Micrococci*”[48]. However, *Staphylococcus (S.) lentus*, *S. sciuri*, and *S. vitulus* can give a positive Microdase reaction. The impact is probably minimal because in an evaluation of CNS infections, 86 cultures revealed one *S. sciuri* and no *S. lentus* or *S. vitulus* [49]. The catalase, Kovacs indole, modified oxidase, and oxidase tests are standard testing agents for the identification of bacteria [43]. The Phadebact D test was found in one study to be 100% effective in identifying Group D *Streptococcus* [50]. Unfortunately, only 80% of *Enterococcus* can be identified by group D antigen testing [43].

Statistical Analysis

Data analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 12.0. A .05 level of probability was chosen to indicate statistical significance. Descriptive statistics (frequencies and counts) were used to summarize and describe the variables in the study. Independent sample chi-square tests were used to evaluate the relationship between bacterial colony counts and surgical service, the time of day when cultures were collected, location of the telephone in the perioperative environment, surgical case number, and OR temperature and OR humidity at the time of sampling.

Findings

Of the six bacteria we attempted to identify, only CNS was found. Additionally, *A. calcoaceticus-baumannii* complex, *P. aeruginosa*, *A. radiobacter/tumefaciens*, *Micrococcus*, *Streptococcus* non-group D, and one unidentified gram-negative rod were found. Table 2 summarizes the types and number of bacterial isolates discovered with comparative separation of

data collection at day one and day two. Only the first culture results for telephones that were double cultured are included in Table 2 to avoid over-representation of isolates.

{Please insert Table 2 Findings Table}

Table 3 summarizes the five variables that remained in the study after the removal of the temperature and humidity variables. The humidity and temperature data were not included due to a lack of standardized measuring instruments. In Table 3, the acronym “BAP” refers to tripticase soy blood agar plates and the number of colonies identified; “CAP” refers to chocolate agar plates; and “MAC” refers to MacConkey agar plates. An absence of colonies at both 24 and 48 hours is indicated by “0,0”. In Table 3 the word “Total” refers to a cumulative total of all colonies identified among the three growth mediums of BAP, CAP and MAC. “CFU/cm²” refers to the number of colony forming units identified per centimeter squared, as determined by a swab path surface area of 28.6875 cm². “Service” refers to the surgical case type that had occurred in the OR just prior to sampling. “Time of Day” refers to whether specimens were collected in the a.m. or p.m. “Culture Location” refers to the area that contained the telephone, which was cultured (i.e., OR Suite or Sub-sterile Room). “Case #” refers to the surgical case sequence in the room during culturing (i.e. 1st, 2nd or 3rd case of the day).

{Please insert Table 3 Summary Table}

We collected the majority of our samples in the a.m. (61.5%) and 38.5% were collected in the p.m. The largest numbers of specimens were obtained from the first surgical case of the day (65.4%), followed by the second case at 30.8%, and the third case at 3.8%. The top five surgical services operating within the rooms where the telephone cultures were obtained were: Orthopedics at 42.3%, Ophthalmology at 15.4%, General Surgery at 11.5%, and Cardiothoracic and Genitourinary Surgery both at 7.7 %. The double cultures from data collection day one and

two revealed CNS of similar quantities and on data collection day two, testing with the Vitek and GPI cards revealed the same Genus and Species, *Staphylococcus epidermidis*. Chi-Square analyses were not significant on any of the variable relationship evaluations. This lack of significance is most likely due to the small sample size in this study. One telephone (specimen #9910 in Table 3) was cultured after the room had been cleaned. This specimen had the second highest number of colony forming units per cm² at 2.16.

Discussion

In our study we were unable to find *S. aureus*, *Enterococcus spp.*, and *E. coli* on telephones in the OR. The inability to find *Enterococcus spp.* may be related to the limitations previously described for the Phadebact D test. Thus, in each of the three isolates that we recorded as non-group D *Streptococcus*, we may have missed possible *Enterococcus*. Additionally, colony counts for bacteria were low in comparison to levels recorded for public school telephones [39], horizontal surfaces in OR rooms (5.86-6.98 CFU/cm²) [27], stethoscopes (158 CFUs) [12], hospital pagers (39-153 CFUs) [51], and telephones in the ICU (7-282 CFU) [38].

The only environmental surface contamination guidelines that we could find were based on the use of RODAC plates. The guidance describes that floors with microbial contamination greater than 50 colonies per plate relate to poor cleanliness [52]. In our study, only four samples exceeded this amount. Those four samples did not contain the isolates of: *Acinetobacter*, *Pseudomonas*, or *Agrobacterium*. Again, our findings were generally low in comparison to other studies. This is largely due to sampling technique variations. For instance, the Yalowitz study swabbed the entire surface of the telephones and would be expected to have higher colony forming units. We only counted bacteria at 24 hours and may have missed some slow growing

bacteria. If an agar plate contained no colonies at 24 hours, this was re-evaluated at 48 hours. Other studies counted bacteria on all plates at 24 and 48 hours [34]. Our inability to find *S. aureus* parallels similar difficulties in another study that were remedied by using broth to support environmental cultures which led to increasing MRSA findings by a factor of two [53]. Similarly, Rafferty and Pancoast (1984) were only able to isolate *S. aureus* twice out of 114 specimens. Our inability to find *E. coli* on telephones is consistent with a study by Rafferty and Pancoast (1984) that found no gram-negative bacteria on 20 telephones in the OR, ICU, Recovery and ER areas [37]. Additionally, *E. coli* was found in the literature review to have a relatively short life span on environmental surfaces [35] [36], which may explain its absence in our study.

The bacterium most frequently isolated in our study was CNS. CNS and *S. aureus* are the most commonly implicated bacteria in surgical site infections, 20% and 14% respectively [3]. Subsequently, CNS are one of the most frequently isolated bacteria in the laboratory [54]. These bacteria are of little virulence [55] but are frequently implicated as the cause of infections in patients who are immunocompromised or have medical implants [56] [57] [58] [59]. A high prevalence of CNS on telephones is consistent with Cozanitis et al. in their finding of CNS on all the telephones that they cultured [38].

Serendipitous Findings

Our results differ from the findings obtained by Rafferty and Pancoast (1984) in that we found three other gram-negative bacteria present on telephones, namely: *A. calcoaceticus-baumannii* complex, *P. aeruginosa*, and *A. radiobacter/tumefaciens*. Similarly, Getchell-White et al. were also able to find *Acinetobacter* and *Pseudomonas* but they were also able to find three isolates of *S. aureus* from 26 telephone cultures. They utilized RODAC impression agar plates

which eliminate the number of times that bacteria are transferred (RODAC plates are a direct transfer technique). Undoubtedly, some of the bacteria remained in our culture swabs and were not accounted for. Had we utilized RODAC plates our bacterial count could have been higher.

The *Acinetobacter* (*A.*) *calcoaceticus-baumannii* complex is made up of four distinctive genotypes yet similar phenotypes - all which cause nosocomial infections[60]. For these reasons, *A. calcoaceticus* and *A. baumannii* will be addressed interchangeably. Both, *A. calcoaceticus*, a Gram-negative, nonfermentative, coccobacillus and *A. baumannii*, a Gram-negative, non motile bacterium can be found as a normal part of skin flora (25%) [61, 62]. *A. calcoaceticus* and *A. baumannii* have been implicated in hospital outbreaks causing patient colonization, infections, and in some cases mortality [60, 61, 63]. *A. calcoaceticus* was found on the skin flora of a respiratory therapist who regularly handled respiratory equipment, thereby causing respiratory tract colonization and infections in endotracheal intubated patients [61]. However, it is *A. baumannii* that has been the culprit in most hospital acquired infections [64]. The fact that *A. baumannii* can survive up to four months on dry surfaces has possible implications in cross contamination of the hospital environment [65]. And, even after new cleaning protocols were implemented in a neurosurgical ICU, isolates of *A. baumannii* were still colonized from patients [66]. In the burn unit of a public teaching hospital, total hospital costs of treating a patient with multidrug-resistant *A. baumannii* (MDRAB) were \$98,575 higher than that of a non-MDRAB patient [67]. Since the advent of using multiple antimicrobial agents in treating nosocomial infections and ongoing developing isolates, the *Acinetobacter* spp. has become more and more resistant to many therapies [62]. For obvious reasons, we need to control the spread of this bacterium before it becomes a super bug. It is likely that our finding of *Acinetobacter* spp. could have been avoided with simple handwashing, surface disinfection and

basic aseptic techniques.

Our finding of *Pseudomonas aeruginosa* (*P. aeruginosa*) is not entirely surprising since this environmental bacteria inhabits soil, water and surfaces with soil or water [68] and has been previously found on telephones in the ICU [34]. *P. aeruginosa* can be a part of the normal flora for some individuals [68] [69]. But, this organism is also an opportunistic pathogen responsible for many severe nosocomial infections such as septicemia, urinary tract infections, pneumonia, and osteochondritis [68]. *Pseudomonas* infections are most commonly found in immunocompromised patients and patients with cystic fibrosis [68] [69]. For those patients who become infected, treatment can be complicated by antibiotic resistance [68]. *Pseudomonas* has been implicated in eight percent of surgical infections [69] and 10 percent of all hospital-acquired infections [68]. In 2001, a CDC investigation of a *Pseudomonas* outbreak revealed that defective bronchoscopes could be a reservoir for infection [70]. A 2003 study by Leibovitz et al. found 34 percent of 50 patients fed with nasogastric tubes were colonized with *P. aeruginosa* in their oral pharynx [71]. Thus, both nasogastric tubes and bronchoscopes have been implicated as fomites or reservoirs. The oral cavity may be a potential site of growth for pathogenic organisms [71]. The presence in the mouth or throat could explain the transmission to the telephone. Improper hand washing [72] and the handling of an endotracheal tube, oral airway, or some other contaminated object could also have contributed to this transmission.

Agrobacterium radiobacter/tumefaciens is rarely implicated as an infectious agent. Of the 42 patients with *Agrobacterium* infections found in literature by 2003 [73], most involved immunocompromised hosts [73] [74] or had invasive procedures involving the placement of plastic implants such as central lines [73] [75] or prosthetic valve [76]. Despite the antibiotic resistance of *Agrobacterium* [77], infections from this bacteria are rarely fatal [78]. The apparent

affinity of this bacterium toward plastic may explain its presence on a telephone in our study. This bacterium was very slow growing and was seen only as a pinpoint colony at 24 hours.

Lastly, we found *Micrococcus* on telephones in the O.R. “The genus *Micrococcus* is currently composed of three species.” [43] p. 385. “*M. luteus* is the most common micrococcal species found in nature and in clinical specimens” [43] p.385. *Micrococci* are considered to be opportunistic pathogens for immunocompromised hosts [43] and have been implicated in endocarditis [79] and central venous catheter infections [80].

Recommendations for improvement

During the collection of temperature and humidity data within the surgical rooms, we utilized gauges already present in rooms. Because no standardized method of measurement was used, these data were removed from our analysis. Future studies could utilize a portable device to ensure standardized measurement. A larger sample size would help to validate data and provide a basis for inferential statistics. Additionally, specimen collection may be more efficient with RODAC impression agar plates, due to the advantages of a direct transfer technique. Colonies should be counted at both 24 and 48 hours in an attempt to recognize slow growing bacteria. In our study, morphologic examination was conducted at 24 hours. However, the separation of colonies based on morphologic examination is much easier at 48 hours. By that time, hemolytic rings are clearly visible and colonies have had ample time to differentiate themselves. While some CNS have been shown to be methicillin resistant [81] [3] [82], it may be beneficial to determine the frequency of this resistance. Finally, adding broth to support environmental cultures could lead to increased MRSA findings as supported by the study conducted by Boyce et al. [53].

Implications and Conclusion

We are cautious in drawing conclusions based on a convenience sample from only one medical center in which surgical technologists and circulators perform cleaning between cases. Our data may have been impacted by increased workloads. During the time of sampling the number of surgical cases increased 25.7% between the first and second sampling. During the entire month of sampling, there was an increase in the number of surgical cases by 37.9% from the previous year. Additionally, the quantity of bacteria needed to cause disease is unclear. Hinton, Maltman, and Orr were able to show that fresh *Staphylococci* intramuscular injections of 100,000 cells could cause infections in 20% of mice while dried *Staphylococci* injections of 400,000 cells caused infections in 10% of mice [83]. However, our CFUs were low in comparison to the Hinton, Maltman, and Orr (1960) study and thus we are unable to draw conclusions related to disease.

Our study does support the need for heightened awareness of cleaning procedures and standard precautions. It seems reasonable to assume that surface contamination in the form of *Acinetobacter* or *Pseudomonas* in the surgical suite is a risk to both staff and patients. While we make a case for cleaning, it must be emphasized that cleaning needs to be done correctly. When bacteria are subjected to sub-lethal levels of disinfectants, they can become resistant to antibiotics [53] [80]. The current guidance by the Association of periOperative Registered Nurses (AORN) for environmental cleaning includes terminally cleaning telephones at the end of the day [18]. Additionally, AORN describes that such cleaning should occur when equipment is visibly soiled. In our study, one telephone (specimen #9910 in Table 3) was cultured after the room had been cleaned (between cases). This specimen had the second highest number of colony forming units per cm² at 2.16. This finding is particularly disturbing and raises the

question: "Should we be cleaning telephones and other objects frequently contacted by hands in the perioperative environment between cases rather than at the end of the day?" More importantly, it is the initial contamination of telephones rather than the cleaning that is concerning. Standard precautions require workers to wear gloves when the possibility of exposure to body fluids exists. Upon removing gloves, hands should be washed [75]. The obvious conundrum for OR personnel follows: I must touch soiled materials, leaving the room decreases positive air pressure and places patients at risk for infection [18] and running water is not available in the surgical suite. A possible solution may include waterless hand-cleaner in surgical suites. Ultimately, the cleanliness of the surgical suite is the responsibility of perioperative nurses [18]. Perioperative managers in concert with Infection Control Officers must ensure that Environmental Protection Agency (EPA) approved hospital disinfectants are both appropriate for emerging resistant bacteria and are being utilized correctly. Close attention must be applied to these key processes and focused to include aseptic principles and standard precautions.

Future research may seek to: measure hand washing compliance in the surgical suite; quantify the number of varying bacteria that can be transferred from surfaces to incisions via gloved hands and cause infection in mice; frequency with which operating room personnel contact surfaces with gloves after performing the function requiring the use of those gloves; and/or quantify contamination of other objects frequently contacted by hands such as door handles or computer keyboards.

{Please insert Figure 1}

References

1. CDC, *National Center for Health Statistics Vital and Health Statistics, Detailed diagnoses and procedures national hospital discharge survey 1994*. Vol. 127. 1997, Hyattsville, MD: Department of Health and Human Services.
2. Weinstein, R.A., *Nosocomial infection update*, E.I. Diseases, Editor. 1998.
3. CDC, *Guideline for prevention of surgical site infection*, in APIC. 1999.
4. Engemann J.J., C.Y., Cosgrove S.E., Fowler V.G., Bronstein M.Z., Trivette S.L., Briggs J.P., Sexton D.J., and K. K.S., *Adverse clinical and economic outcomes attributable to methicillin resistance among patients with Staphylococcus aureus surgical site infection*. Clinical Infectious Diseases, 2003. **36**(5): p. 592-598.
5. Altemeier, W.A., W.R. Culbertson, and R.P. Hummel, *Surgical considerations of endogenous infections--sources, types, and methods of control*. Surg Clin North Am, 1968. **48**(1): p. 227-40.
6. Wiley, A.M. and G.B. Ha'eri, *Routes of infection. A study of using "tracer particles" in the orthopedic operating room*. Clin Orthop, 1979(139): p. 150-5.
7. APIC, *Infection control & applied epidemiology: Principles and practice*. 1996, St. Louis, MO: Mosby.
8. Tortora, G.J., Funke, B. R., & Case, C. L., *Microbiology an introduction*. 1995, Redwood City, CA: The Benjamin Cummings Publishing Co. Inc.
9. Gruendemann, B.J., Mangum, S. S., *Infection Prevention in Surgical Settings*. 2001, Philadelphia, PA: Saunders. 387.
10. Rutala, W.A., *Disinfection and sterilization of patient-care items*. Infection Control and Hospital Epidemiology, 1996. **17**(6): p. 377-384.
11. Scott, E.B., S. F., *The survival and transfer of microbial contamination via cloths, hands and utensils*. Journal of Applied Bacteriology, 1990. **68**: p. 271-278.
12. Marinella, M.A., Pierson, C., & Chenoweh, C., *The stethoscope: A potential source of nosocomial infection*. Arch Intern Med, 1997. **157**: p. 786-790.
13. Larson, E., *A causal link between handwashing and risk of infection? Examination of the evidence*. Infection Control and Hospital Epidemiology, 1988. **9**(1): p. 28-36.
14. Semmelweis, I.P., *The etiology, the concept and the prophylaxis of childbed fever*, in *Translation by Murphy, F. B. 1981*. 1861: Pest, Hungary.
15. Bischoff W.E., R.T.M., Sessler C.N., Edmond M.B., Wenzel R.P., *Handwashing compliance by health care workers: The impact of introducing an accessible, alcohol-based hand antiseptic*. Archives of Internal Medicine, 2000. **160**(7): p. 1017-21.
16. Tait, A.R. and D.B. Tuttle, *Preventing perioperative transmission of infection: a survey of anesthesiology practice*. Anesth Analg, 1995. **80**(4): p. 764-9.
17. Pittet D, S.F., Hugonnet S, Akakpo C, Souweine B, Clergue F., *Hand-cleansing during postanesthesia care*. Anesthesiology, 2003. **99**(3): p. 519-520.
18. A.O.R.N., *Standards, Recommended Practices, and Guidelines*. 2004, Denver Colorado: AORN. 398.
19. Davies, J., *Inactivation of antibiotics and the dissemination of resistance genes*. Science, 1994. **264**(5157): p. 375-82.
20. Rice, L.B., *Bacterial monopolists: the bundling and dissemination of antimicrobial resistance genes in gram-positive bacteria*. Clin Infect Dis, 2000. **31**(3): p. 762-9. Epub 2000 Oct 04.

21. Higgins, N.P., *Death and transfiguration among bacteria*. Trends Biochem Sci, 1992. **17**(6): p. 207-11.
22. Cohen, M.L., E.S. Wong, and S. Falkow, *Common R-plasmids in Staphylococcus aureus and Staphylococcus epidermidis during a nosocomial Staphylococcus aureus outbreak*. Antimicrob Agents Chemother, 1982. **21**(2): p. 210-5.
23. Trieu-Cuot, P., E. Derlot, and P. Courvalin, *Enhanced conjugative transfer of plasmid DNA from Escherichia coli to Staphylococcus aureus and Listeria monocytogenes*. FEMS Microbiol Lett, 1993. **109**(1): p. 19-23.
24. Perry, S.M. and W.P. Monaghan, *The prevalence of visible and/or occult blood on anesthesia and monitoring equipment*. Aana J, 2001. **69**(1): p. 44-8.
25. Devine, J., Cooke, R. P. D., & Wright, E. P., *Is methicillin-resistant Staphylococcus aureus (MRSA) contamination of ward-based computer terminals a surrogate marker for nosocomial MRSA transmission and handwashing compliance?* Journal of Hospital Infection, 2001(48): p. 72-75.
26. Dharan, S., Mourouga, P., Copin, P., Bessmer, G., Tschanz, B., & Pittet, D., *Routine disinfection of patients' environmental surfaces. Myth or reality?* Journal of Hospital Infection, 1999. **42**: p. 113-117.
27. Weber, D.O., Gooch, J. J., Walter, R. W., Britt, E. M., & Kraft, R. O., *Influence of operating room surface contamination on surgical wounds*. Arch Surg, 1976. **111**(484-488).
28. Boyce, J.M., *Patterns of methicillin-resistant Staphylococcus aureus prevalence*. Infect Control Hosp Epidemiol, 1991. **12**(2): p. 79-82.
29. Porwancher, R., et al., *Epidemiological study of hospital-acquired infection with vancomycin-resistant Enterococcus faecium: possible transmission by an electronic ear-probe thermometer*. Infect Control Hosp Epidemiol, 1997. **18**(11): p. 771-3.
30. Noskin, G.A., et al., *Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces*. Infect Control Hosp Epidemiol, 1995. **16**(10): p. 577-81.
31. Neely, A.N. and M.P. Maley, *Survival of enterococci and staphylococci on hospital fabrics and plastic*. J Clin Microbiol, 2000. **38**(2): p. 724-6.
32. Wendt, C., et al., *Survival of vancomycin-resistant and vancomycin-susceptible enterococci on dry surfaces*. J Clin Microbiol, 1998. **36**(12): p. 3734-6.
33. Bonilla, H.F., M.J. Zervos, and C.A. Kauffman, *Long-term survival of vancomycin-resistant Enterococcus faecium on a contaminated surface*. Infect Control Hosp Epidemiol, 1996. **17**(12): p. 770-2.
34. Getchell-White, S.I., L.G. Donowitz, and D.H. Groschel, *The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of Acinetobacter calcoaceticus*. Infect Control Hosp Epidemiol, 1989. **10**(9): p. 402-7.
35. Fryklund, B., K. Tullus, and L.G. Burman, *Survival on skin and surfaces of epidemic and non-epidemic strains of enterobacteria from neonatal special care units*. J Hosp Infect, 1995. **29**(3): p. 201-8.
36. Smith, S.M., R.H. Eng, and F.T. Padberg, Jr., *Survival of nosocomial pathogenic bacteria at ambient temperature*. J Med, 1996. **27**(5-6): p. 293-302.
37. Rafferty, K.M.P., S. J., *Brief report: Bacteriological sampling of telephones and other hospital staff hand-contact objects*. Infection Control, 1984. **5**(11): p. 533-535.
38. Cozanitis, D.A., Grant, J., & Makela, P., *Bacterial contamination of telephones in an*

- intensive care unit. Anaesthesist*, 1978. **27**: p. 439-442.
39. Yalowitz, M.B., I., *The recovery of bacteria from the handpiece of a high school telephone*. *Journal of Environmental Health*, 2003. **65**(6): p. 18-20.
 40. Rusin, P., S. Maxwell, and C. Gerba, *Comparative surface-to-hand and fingertip-to-mouth transfer efficiency of gram-positive bacteria, gram-negative bacteria, and phage*. *J Appl Microbiol*, 2002. **93**(4): p. 585-92.
 41. Harbarth, S., et al., *Interventional study to evaluate the impact of an alcohol-based hand gel in improving hand hygiene compliance*. *Pediatr Infect Dis J*, 2002. **21**(6): p. 489-95.
 42. Isenberg, H.D., *Clinical microbiology procedures handbook*, ed. A.S.f. Microbiology. Vol. 1. 1994.
 43. Murray, P.R., Baron, E.J., Pfaller, M.A., Jorgensen, J.H., and Tenover, R.H., *Manual of Clinical Microbiology*. 8 ed, ed. P.R. Murray. Vol. 1. 2003, Washington D.C.: ASM Press.
 44. Remel, *Staphaurex Plus ZL33/34*. 2002. p. Product insert.
 45. Wilkerson, M., et al., *Comparison of five agglutination tests for identification of Staphylococcus aureus*. *J Clin Microbiol*, 1997. **35**(1): p. 148-51.
 46. bioMerieux, *Gram-Negative Identification+ Card for in vitro diagnostic use Pininsert (Rev 0498 / I)*. 1999, bioMerieux Vitek Inc.
 47. Faller, A. and K.H. Schleifer, *Modified oxidase and benzidine tests for separation of staphylococci from micrococci*. *J Clin Microbiol*, 1981. **13**(6): p. 1031-5.
 48. Baker, J.S., *Comparison of various methods for differentiation of staphylococci and micrococci*. *J Clin Microbiol*, 1984. **19**(6): p. 875-9.
 49. Lang, S., et al., *The genomic diversity of coagulase-negative staphylococci associated with nosocomial infections*. *J Hosp Infect*, 1999. **43**(3): p. 187-93.
 50. Burdack, N.M., et al., *Group identification of streptococci. Evaluation of three rapid agglutination methods*. *Am J Clin Pathol*, 1981. **76**(6): p. 819-22.
 51. Singh, D., Kaur, H., Gardner, W. G., & Treen, L. B., *Bacterial contamination of hospital papers*. *Infection Control and Hospital Epidemiology*, 2002. **23**(5): p. 274-276.
 52. Pryor, A.K., Veslely, D., & Shaffer, J. G., *Cooperative microbial surveys of surfaces in hospital patient rooms*. *Health Lab Sciences*, 1967. **4**: p. 153-159.
 53. Boyce, J.M., et al., *Environmental contamination due to methicillin-resistant Staphylococcus aureus: possible infection control implications*. *Infect Control Hosp Epidemiol*, 1997. **18**(9): p. 622-7.
 54. Patrick, C.C., *Coagulase-negative staphylococci: pathogens with increasing clinical significance*. *J Pediatr*, 1990. **116**(4): p. 497-507.
 55. Rothrock, J.C., *Alexander's care of the patient in surgery*. 12th ed. ed. 2003, St. Louis: Mosby Inc.
 56. Cunha Mde, L., et al., *[Clinical significance of coagulase-negative staphylococci isolated from neonates]*. *J Pediatr (Rio J)*, 2002. **78**(4): p. 279-88.
 57. Lesseva, M., *Central venous catheter-related bacteraemia in burn patients*. *Scand J Infect Dis*, 1998. **30**(6): p. 585-9.
 58. al-Rashdan, A., R. Bashir, and F.A. Khan, *Staphylococcus capitis causing aortic valve endocarditis*. *J Heart Valve Dis*, 1998. **7**(5): p. 518-20.
 59. Rupp, M.E. and G.L. Archer, *Coagulase-negative staphylococci: pathogens associated with medical progress*. *Clin Infect Dis*, 1994. **19**(2): p. 231-43; quiz 244-5.
 60. Gerner-Smidt, P., *Ribotyping of the Acinetobacter calcoaceticus-Acinetobacter*

- baumannii* complex. J Clin Microbiol, 1992. **30**(10): p. 2680-5.
61. Buxton, A.E., Anderson, R. L., Werdegar, D, Atlas, E., *Nosocomial respiratory tract infection and colonization with Acinetobacter calcoaceticus*. The American Journal of Medicine, 1978. **65**: p. 507-513.
62. Borgmann, S., et al., *Metallo-beta-lactamase expressing multi-resistant Acinetobacter baumannii transmitted in the operation area*. J Hosp Infect, 2004. **57**(4): p. 308-15.
63. Scerpella, E.G., Wanger, A. R., Armitige, L., Anderlini, P., & Ericsson, C. D., *Nosocomial outbreak caused by a multiresistant clone of Acinetobacter baumannii: Results of the case-control and molecular epidemiologic investigations*. Infect Control Hosp Epidemiol, 1993. **16**(2): p. 92-97.
64. Bergogne-Berezin, E., and Towner, K. J., *Acinetobacter spp. as nosocomial pathogens: Microbiological, clinical, and epidemiological features*. Clinical Microbiology Reviews, 1996. **9**(2): p. 148-165.
65. Wendt, C., Dietze, B., Dietz, E., & Ruden, H., *Survival of Acinetobacter baumannii on dry surfaces*. Journal of Clinical Microbiology, 1997. **35**(6): p. 1394-1397.
66. Denton, M., et al., *Role of environmental cleaning in controlling an outbreak of Acinetobacter baumannii on a neurosurgical intensive care unit*. J Hosp Infect, 2004. **56**(2): p. 106-10.
67. Wilson, S.J., Knipe, C. J., Zieger, M. J., Gabehart, K. M., Goodman, J. E., Volk, H. M., and Sood, R., *Direct costs of multidrug-resistant Acinetobacter baumannii in the burn unit of a public teaching hospital*. APIC, 2004. **32**(6): p. 342-344.
68. Todar, K.U.o.W.-M.D.o.B., *Todar's Online Textbook of Bacteriology*. 2004, Kenneth Todar University of Wisconsin-Madison Dept. of Bacterology.
69. Van Delden, C.I., B. H., *Cell to Cell signaling and Pseudomonas aeruginosa Infections*. Emerging Infectious Diseases, 1999. **4**(4).
70. CDC, *Notice to Readers: Pseudomonas aeruginosa Infections Associated with Defective Bronchoscopes*. 2001, CDC.
71. Leibovitz, A., Dan, M., Zinger, J., Carmeli, Y., Habot, B., Segal, R., *Pseudomonas aeruginosa and the Oropharyngeal Ecosystem of Tube-Fed Patients*. Emerging Infectious Diseases, 2003. **9**(8).
72. Foca M, J.K., Whittier S, Della Latta P, Factor S, Rubenstein D, Saiman L., *Endemic Pseudomonas aeruginosa infection in a neonatal intensive care unit*. N Engl J Med, 2000. **343**(10): p. 695-700.
73. Amaya, R.A. and M.S. Edwards, *Agrobacterium radiobacter bacteremia in pediatric patients: case report and review*. Pediatr Infect Dis J, 2003. **22**(2): p. 183-6.
74. Jankauskiene, A., et al., *Peritonitis caused by Agrobacterium tumefaciens in a child on peritoneal dialysis*. Nephrol Dial Transplant, 2003. **18**(11): p. 2456-7.
75. Edmond, M.B., et al., *Agrobacterium radiobacter: a recently recognized opportunistic pathogen*. Clin Infect Dis, 1993. **16**(3): p. 388-91.
76. Plotkin, G.R., *Agrobacterium radiobacter prosthetic valve endocarditis*. Ann Intern Med, 1980. **93**(6): p. 839-40.
77. Hulse, M., S. Johnson, and P. Ferrieri, *Agrobacterium infections in humans: experience at one hospital and review*. Clin Infect Dis, 1993. **16**(1): p. 112-7.
78. Paphitou, N.I. and K.V. Rolston, *Catheter-related bacteremia caused by Agrobacterium radiobacter in a cancer patient: case report and literature review*. Infection, 2003. **31**(6): p. 421-4.

79. Seifert, H., M. Kaltheuner, and F. Perdreau-Remington, *Micrococcus luteus endocarditis: case report and review of the literature*. Zentralbl Bakteriол, 1995. **282**(4): p. 431-5.
80. Oudiz, R.J., et al., *Micrococcus-associated central venous catheter infection in patients with pulmonary arterial hypertension*. Chest, 2004. **126**(1): p. 90-4.
81. Hanssen, A.M., G. Kjeldsen, and J.U. Sollid, *Local variants of Staphylococcal cassette chromosome mec in sporadic methicillin-resistant Staphylococcus aureus and methicillin-resistant coagulase-negative Staphylococci: evidence of horizontal gene transfer?* Antimicrob Agents Chemother, 2004. **48**(1): p. 285-96.
82. Mohanty, S.S. and P.R. Kay, *Infection in total joint replacements. Why we screen MRSA when MRSE is the problem?* J Bone Joint Surg Br, 2004. **86**(2): p. 266-8.
83. Hinton, N.A., J.R. Maltman, and J.H. Orr, *The effect of desiccation on the ability of Staphylococcus pyogenes to produce disease in mice*. Am J Hyg, 1960. **72**: p. 343-50.

Figure 1

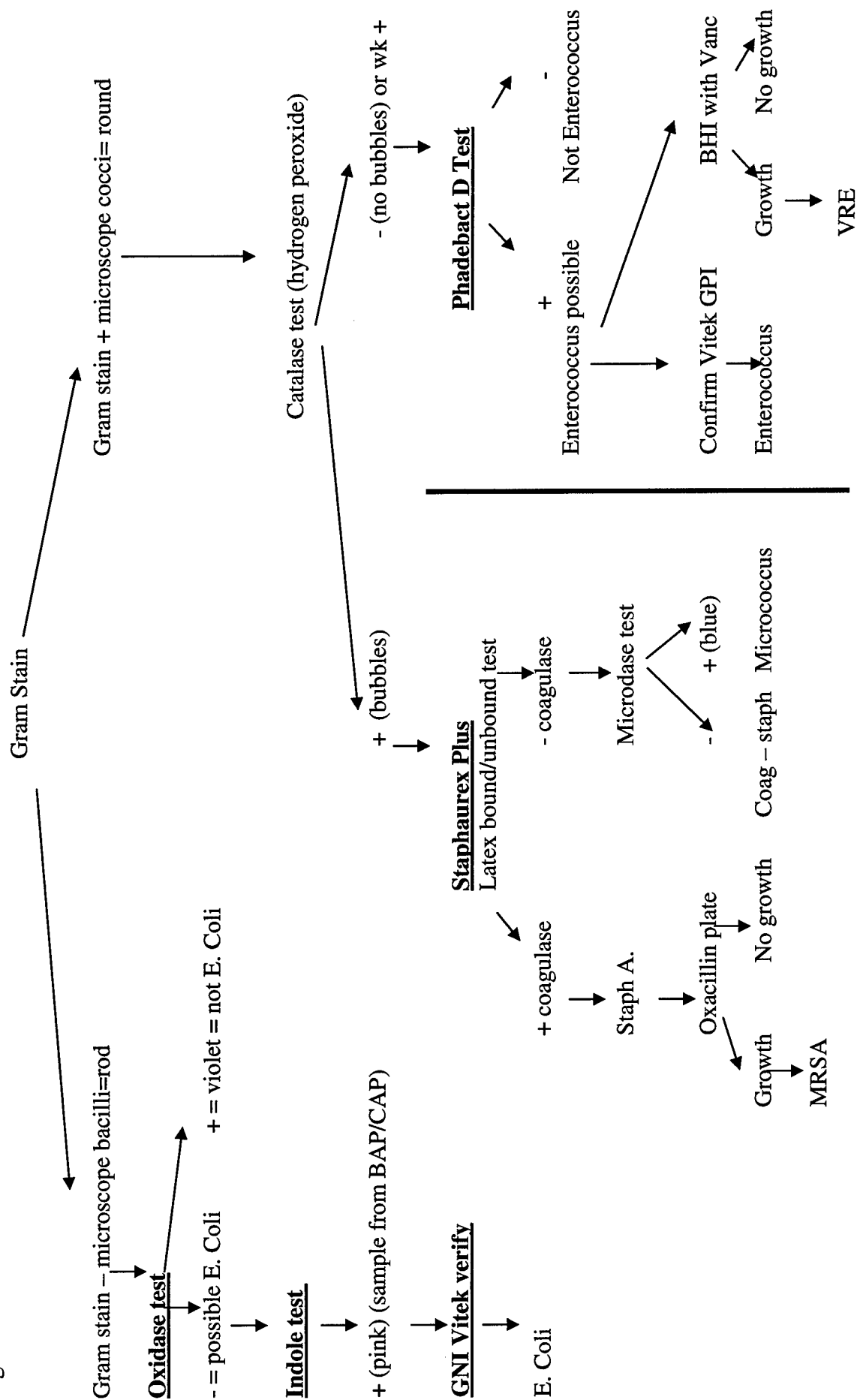


Table 1 Bacteria Survival on Surfaces

Type of Bacteria	Bacteria Survival Time on Hands	Bacteria Survival Time on Inanimate Surfaces	Author	Year	Type of Study
<i>Enterococcus spp</i>	Gloved and ungloved hands 60 min [29]	1. Plastic = 68-90 days [30]	Neely and Maley	2000	Quasi-experimental
		2. Telephone = 60 min [29]	Noskin, et al.	1995	Quasi-experimental
		3. PVC = 1 wk to 4 months [31]	Wendt, et al.	1998	Quasi-experimental
VRE	Gloved and ungloved hands 60 min [29]	1. Counter = 58 days [32]	Bonilla, Zervos, Kauffman	1996	Quasi-experimental
		2. Telephone = 60 min [29]	Noskin, et al.	1995	Quasi-experimental
<i>S. aureus</i>	Known skin flora.	3. PVC = 1 wk to 4 months [31]	Wendt, et al.	1998	Quasi-experimental
		1. Plastic = 22- >90 days [30]	Neely and Maley	2000	Quasi-experimental
		2. Formica = 2 days [33]	Getchell-White et al.	1989	Quasi-experimental
MRSA	Known to survive as skin flora particularly in nares.	1. Plastic = 40-51 days [30]	Neely and Maley	2000	Quasi-experimental
<i>E. coli</i>	6 min [34]	1. Glass = 15 min [34]	Fryklund, Tullus, Burman	1995	Experimental
CNS	Known skin flora.	2. Polystyrene tubes = 3 days [35]	Smith, Eng, and Padberg	1996	Quasi-experimental
		1. Plastic = 41->90 days [30]	Neely and Maley	2000	Quasi-experimental

Table 2 Findings Table

Type of Bacteria	# of Isolates Specimen Collection Day 1	# of Isolates Specimen Collection Day 2
<i>Acinetobacter calcoaceticus-baumannii</i> complex	0	1
<i>Pseudomonas aeruginosa</i>	0	1
<i>Agrobacterium radiobacter/tumefaciens</i>	0	1
Coagulase negative <i>staphylococcus</i>	18	25
<i>Micrococcus</i>	0	2
<i>Escherichia. coli</i>	0	0
Vancomycin-resistant <i>Enterococcus</i>	0	0
Methicillin-resistant <i>Staphylococcus aureus</i>	0	0
<i>Staphylococcus aureus</i>	0	0
<i>Streptococcus</i> non-group D	1	2
Unidentified gram negative rod (ruled out <i>Acinetobacter</i> , ESBL, <i>Bacillus anthracis</i> , and <i>Haemophilus</i>)	1	0

Table 3 Variable Summary Table

Day 1 Specimen Collection	BAP	CAP	MAC	Total	CFU/cm ²	Service	Time of Day	Culture Location	Case #
9901	25	26	0,0	51	1.78	GEN	a.m.	OR Suite	2
9902	5	3	0,0	8	0.28	NEURO	a.m.	Sub-sterile	2
9903 DBL	3	3	0,0	6	0.21	PLAST	p.m.	OR Suite	1
9904 DBL	6	7	0,0	13	0.45	PLAST	p.m.	OR Suite	1
9905	8	6	0,0	14	0.49	GEN	p.m.	Sub-sterile	2
9906	14	3	0,0	17	0.59	ORTHO	p.m.	OR Suite	1
9907	0,0	0,0	0,0	0	0.00	EYE	a.m.	OR Suite	2
9908 Control	0,0	0,0	0,0	0	0.00	N/A	N/A	N/A	N/A
9909	1	1	0,0	2	0.07	ORTHO	p.m.	OR Suite	2
9910 ^(a)	47	15	0,0	62	2.16	ORTHO	p.m.	OR Suite	1
9911	18	4	0,0	22	0.77	CT	p.m.	OR Suite	3
9912	31	19	0,0	50	1.74	EYE	p.m.	OR Suite	1
9913	30	13	0,0	43	1.50	OMF	p.m.	OR Suite	1
Day 2 Specimen Collection	BAP	CAP	MAC	Total	CFU/cm ²	Service	Time of Day	Culture Location	Case #
9918	6	9	0,0	15	0.52	ORTHO	a.m.	Sub-sterile	1
9919	20	2	0,0	22	0.77	ENT	a.m.	OR Suite	1
9920 Control	0,0	0,0	0,0	0	0.00	N/A	N/A	N/A	N/A
9921	1	0,0	0,0	1	0.03	EYE	a.m.	OR Suite	2
9922	27	10	0,0	37	1.29	GEN	a.m.	Sub-sterile	1
9923 DBL	3	3	0,0	6	0.21	ORTHO	a.m.	OR Suite	1
9924 DBL	5	0,0	0,0	5	0.17	ORTHO	a.m.	OR Suite	1
9925	6	0,0	0,0	6	0.21	EYE	a.m.	OR Suite	1
9926	17	2	0,0	19	0.66	ORTHO	a.m.	OR Suite	1
9927	53	6	0,0	59	2.06	ORTHO	a.m.	OR Suite	1
9928	7	3	0,0	10	0.35	GU	a.m.	OR Suite	1
9929	1	1	0,0	2	0.07	GU	a.m.	OR Suite	2
9930	19	9	0,0	28	0.98	ORTHO	a.m.	OR Suite	1
9931	28	35	0,0	63	2.20	ORTHO	a.m.	OR Suite	2
9932	7	9	1,1	17	0.59	ORTHO	a.m.	OR Suite	1
9933	24	6	0,0	30	1.05	CT	a.m.	OR Suite	1
9934	11	6	0,0	17	0.59	ORTHO	a.m.	OR Suite	1

Note. Specimen Collection was divided among two days that were separated by 19 days.

1. DBL refers to sequential culturing of the same telephone during the same time period.

2. Control refers to the one control culture that was randomly selected for each culture batch.

3. BAP= blood agar plate

4. CAP= chocolate agar plate

5. MAC= MacConkey agar plates

6. CFU/cm² refers to the number of colony forming units identified per centimeter squared.

7. ^(a)= room cleaned prior to swab.